

# Neuropsychological consequences of right thalamic haemorrhage: Case study and review

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## Abstract

The neuropsychological performance of a right-handed man is examined following haemorrhage from the anterior sections of the right thalamus. A pattern of temporally graded retrograde amnesia, global anterograde amnesia, impaired short-term memory, behavioural changes, and severe executive deficits were identified. The deficits evident in this case are discussed in reference to existing neuropsychological literature regarding the consequences of thalamic infarction. It is proposed that damage to the anterior thalamic nuclei results in a frontal dysexecutive syndrome and that such a dysexecutive syndrome can explain the neuropsychological deficits observed in this case. © 2002 Elsevier Science (USA). All rights reserved.

*Keywords:* Thalamic haemorrhage; Dysexecutive syndrome

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## 1. Introduction

Damage to thalamic nuclei has consistently been found to induce neuropsychological impairments. There are reports of memory disturbances (Baumgartner & Regard, 1993; Brown, Kieran, & Patel, 1989; Calabrese, Haptops, Markowitsch, & Gehlen, 1993; Clarke et al., 1994; Hankey & Stewart-Wynne, 1988; Haut, Young, & Cutlip, 1995; Lisovski et al., 1993; Parkin, Rees, Hunkin, & Rose, 1994a; Pepin & Auray-Pepin, 1993; Peru & Fabbro, 1997; Sala, Spinnler, & Venneri, 1997; Sodeyama, Tamaki, & Sugishita, 1995; Speedie & Heilman, 1982, 1983; Van Der Werf et al., 1999), frontal disinhibition (Bogousslavsky et al., 1988), dysarthria (Ackerman, Ziegler, & Petersen, 1993), dysphasia (Karussis, Leker, & Abramsky, 2000; Kumar, Masih, & Pardo, 1996; Lisovski et al., 1993), movement disorders (Lee & Marsden, 1994), thought disorders (Chatterjee et al., 1997), ideomotor apraxia (Nadeau, Roeltgen, Sevush, Ballinger, & Watson, 1994), and prosopoaffective agnosia (Vuilleumier, Ghika-Schmid, Bogousslavsky, Assal, & Regli, 1998) arising from thalamic infarcts and haemorrhages. However, to date a consistent clinical picture of a

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“thalamic syndrome” has not emerged, although most conceptions of a thalamic syndrome include elements of an aphasic and amnesic disorder.

There appears to be considerable uncertainty surrounding the cause of memory deficits arising from thalamic damage. One hypothesis suggests that thalamic amnesia represents a “pure” memory disorder, indicating that the thalamus may be a central structure involved in memory processes in the same manner that the medial temporal lobe is implicated (Calabrese et al., 1993; Haut et al., 1995; Van Der Werf, Witter, Uylings, & Jolles, 2000). Alternately, it is suggested that thalamic amnesia represents a disconnection syndrome as opposed to a pure amnesia, such that the amnesia evidenced is the result of loss of information flow between anatomical regions involved in memory (Bogousslavsky et al., 1988; Clarke et al., 1994; Speedie & Heilman, 1982, 1983). A third possibility is the hypothesis that thalamic amnesia represents a frontal-lobe “amnesia”. Such a frontal amnesia is argued to result from the disconnection of the frontal lobes from medial temporal lobe structures, such as the hippocampus. The resulting amnesia is described as being similar to the frontal-lobe dysexecutive syndrome and represents impairments to memory processing due to a loss of frontal-lobe regulation (Pepin & Auray-Pepin, 1993; Van Der Werf et al., 2000).

Further complexity is added to the concept of a thalamic syndrome with research indicating that the pattern of impairment varies according to the thalamic hemisphere or the thalamic nuclei that are damaged. Left thalamic damage has been associated with verbal anterograde amnesia (Brown et al., 1989; Hankey & Stewart-Wynne, 1988; Lisovoski et al., 1993; Parkin et al., 1994a; Sodeyama et al., 1995; Speedie & Heilman, 1982), intact short-term memory (Brown et al., 1989), long-term anterograde and retrograde amnesia for verbal and visual information (Clarke et al., 1994), dysphasia (Karussis et al., 2000; Kumar et al., 1996; Nadeau et al., 1994; Sodeyama et al., 1995), loss of spontaneity (Kumar et al., 1996), ideomotor apraxia (Nadeau et al., 1994), frontal-lobe dysfunction (Pepin & Auray-Pepin, 1993), and an absence of frontal-lobe compromise (Parkin et al., 1994a). In contrast, damage to the right thalamus has been associated with visuospatial anterograde amnesia (Hanlon, Dobkin, Handler, Ramirez, & Cheska, 1992; Speedie & Heilman, 1983), global anterograde, and retrograde amnesia (Sala et al., 1997), frontal disinhibition (Bogousslavsky et al., 1988; Hashimoto, Yoshida, & Tanaka, 1995), a “dysexecutive” syndrome (Van Der Werf et al., 1999), neglect (Karussis et al., 2000), and anosognosia (Karussis et al., 2000). Further studies have suggested that unilateral thalamic damage may produce bilateral neuropsychological deficits as opposed to unilateral deficits, such deficits being reported as bilateral fronto-temporal impairments (Baumgartner & Regard, 1993).

The inconsistencies evident in studies of unilateral thalamic damage are also evident in studies of specific thalamic nuclei. Chung et al. (1996) in a study of 175 consecutive cases of thalamic haemorrhage determined that anteromedial thalamic haemorrhages were associated with acute confusion and behavioural changes. Posteromedial haemorrhages were associated with severe memory disturbances. Posterolateral haemorrhages were associated with hemi-neglect in the right side and dysphasia in the left side, and dorsal haemorrhages were associated with motor disturbances (Chung et al., 1996). Infarcts localised to the medial thalamic nuclei have been associated with severe anterograde amnesia (Brown et al., 1989; Calabrese et al., 1993; Parkin et al., 1994a; Speedie & Heilman, 1982, 1983) and both intact frontal-lobe functions (Parkin et al., 1994a) and impaired frontal-lobe functions (Speedie & Heilman, 1982, 1983). Infarcts to the anterior thalamic nuclei have been associated with symptoms of frontal-lobe impairment (Clarke et al., 1994; Hashimoto et al., 1995), anterograde amnesia (Clarke et al., 1994; Ebert, Vinz, Görtler, Wallesch, & Herrmann, 1999; Hankey & Stewart-Wynne, 1988; Lisovoski et al., 1993), impaired short-term memory capacity (Ebert et al., 1999), impaired attention and concentration (Ebert et al., 1999), and adynamia and loss of affective expression

(Lisovoski et al., 1993). Infarcts to lateral thalamic nuclei are associated with ideomotor apraxia (Nadeau et al., 1994), dysphasia (Nadeau et al., 1994; Sodeyama et al., 1995), anterograde amnesia (Nadeau et al., 1994; Sodeyama et al., 1995), and hypomania and prosopoaffective agnosia (Vuilleumier et al., 1998).

While the majority of researchers indicate that language and memory disturbances are key features of thalamic damage, a number of studies indicate that such memory disturbances may not be the predominant clinical feature of thalamic damage (Bogousslavsky et al., 1988; Van Der Werf, 2000). In this paper, a case study is presented in which memory disturbances are a feature of thalamic damage however, the key clinical presentation is of a frontal dysexecutive syndrome with subsequent memory impairments. It is argued further that in contrast to representing a medial temporal lobe amnesia, the amnesia evident in this case has greater similarity to the memory disturbances seen following discrete frontal-lobe damage, such as that evidenced following rupture of the anterior communicating artery aneurysm.

## 2. Case study

### 2.1. History

“P.D.” is a 59-year-old single man who was incapable of providing a clear and coherent personal history. Under conditions of highly directive questioning, he reported leaving school at 14 years of age and, subsequently, undertaking two years training at a night school to become an electrician, but reported that he did not qualify as an electrician. On the basis of self-report, P.D.’s occupational history consisted predominantly of seasonal itinerant unskilled labour. P.D. reported as being married, having 2 children but indicated that his wife and children had left him some 30 years ago and that he had no contact with his family. He displayed impoverished recall of his personal history since the 1980s being unable to provide clear details of occupation, residence or other personal information.

P.D. suffered a large right-sided basal ganglia infarction, seven months prior to neuropsychological examination. Medical records indicate that detection of this infarct was secondary to his primary admission for acute myocardial infarction. CT scans conducted seven months prior to neuropsychological assessment indicated a 6 cm diameter right-sided haematoma, resulting from a thalamic haemorrhage (Fig. 1). Anatomical mapping indicated the point of origin of the haemorrhage to be in the right anterior-ventral thalamus (Haines, 1995). The haematoma extended into the right frontal-lobe and the anterior right temporal lobe causing effacement of the anterior right lateral ventricle, a slight dilation of the left lateral ventricle, as well as a midline shift of less than 1 cm. Repeat CT scans over a one-month period subsequent to initial onset indicated no change to the size or severity of the haematoma.

### 2.2. Presentation

P.D. presented as a right-hand dominant man who displayed limited insight into any of the difficulties that he experienced. He denied the presence of cognitive problems. He remained socially appropriate at all times during the assessment however, medical reports indicate a history of sexually inappropriate behaviours displayed during his admission. He presented as unkempt in appearance with a lack of appropriate self-care being evident. His speech was markedly slowed and he displayed a moderate level of oral dyspraxia. His motor behaviour displayed evidence of mild bilateral motor dyspraxia. In mood, he presented as consistently euthymic, but displayed mild blunting of affective range. In addition, he presented as moderately adynamic, displaying considerable difficulty in self-initiation of behaviours.

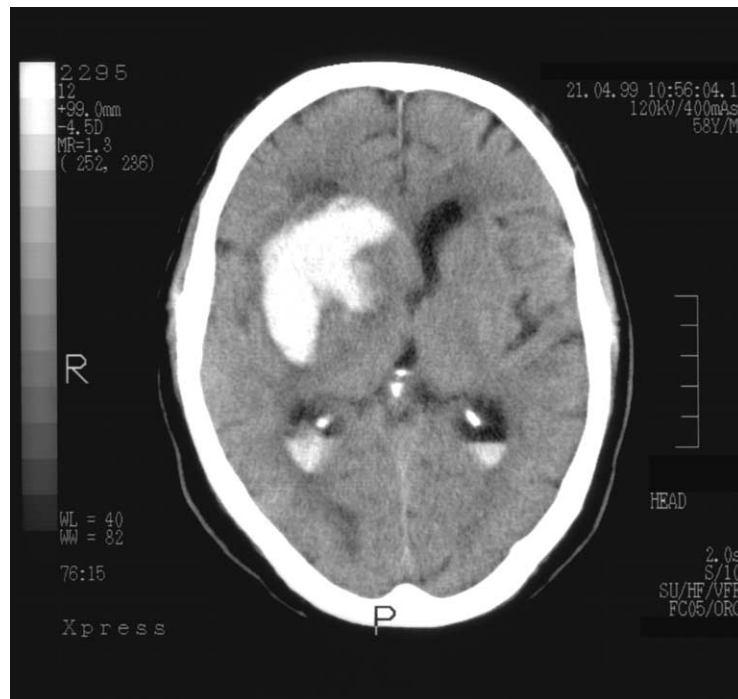


Fig. 1. CT scan of the brain without contrast performed 6 days after admission.

### 2.3. Neuropsychological assessment

Seven months following admission to hospital, P.D. underwent comprehensive neuropsychological assessment. He was fully cooperative with assessment at all times, but displayed no insight into cognitive deficits or into difficulties with tasks as he experienced them (see Table 1).

### 2.4. General cognitive functioning

P.D.'s general intellectual abilities were measured using the Wechsler Adult Intelligence Scale—Revised (WAIS-R). He attained a full-scale IQ of 80, a verbal IQ of 83 and a prorated performance IQ (excluding picture arrangement) of 76. On the basis of best subtest performance (Lezak, 1995), P.D. was estimated to have a pre-morbid intellectual ability in the average range, indicating a significant decline in general intellectual abilities.

### 2.5. Temporal and personal orientation

P.D. displayed marked disorientation for time and place and partial disorientation for persons. He was able to report his name and date of birth, but was incapable of accurately reporting his age. He indicated the correct month, but the incorrect day of the month and indicated that he believed the year to be 9 years prior to the date he was assessed. When asked to name the current and erstwhile Prime Minister of Australia, he was unable to report the current Prime Minister, but reported the erstwhile Prime Minister (who lost an election 4 years, prior to the assessment) and the Prime Minister before that. Thus, while the sequencing of this historical information was correct, he appeared unable to recall historical information about events that occurred in the preceding 4 years.

Table 1  
Neuropsychological test results

Test	Raw score	Percentile
<i>WAIS-R</i>		
FIQ	80	
VIQ	83	
PIQ (prorated)	76	
<i>WMS-III</i>		
Auditory immediate memory	77	6
Visual immediate memory	65	1
Immediate memory	65	1
Auditory delayed memory	74	4
Visual delayed memory	65	1
Auditory recognition delayed	90	25
General memory	70	2
Working memory	69	2
<i>RAVLT</i>		
Trial 1	4	7
Trial 2	7	21
Trial 3	7	5
Trial 4	6	0.8
Trial 5	8	8
Total	32	3
List B	5	42
List A recall	4	4
List A recognition	9	18
<i>Complex figure of rey</i>		
Copy	32	42
Recall (3 min delay)	9	5
Boston naming test	59	84
<i>Controlled oral word association test</i>		
F		
A	7	7
S	5	8
Total	7	5
	19	4
Category fluency	8	3
<i>Trail making test</i>		
Part A	195 s	0.1
Part B	Incomplete	0.1
<i>Stroop test (24 item)</i>		
Colour	28 s, 0 error	0.1
Word	65 s, 1 error	0.1
Colour-word incongruent	115 s, 8 errors	0.1

## 2.6. Memory functioning

P.D.'s memory and learning abilities were assessed using the Wechsler Memory Scale III (WMS-III), the Complex Figure of Rey recall trial, and the Rey Auditory Verbal Learning Test (RAVLT). P.D.'s performances on the WMS-III indicated severe impairments to auditory immediate, auditory delayed, visual immediate, visual delayed, immediate, general memory, and working memory indices.

P.D. displayed reduced immediate memory functions, performing at an impaired level on auditory/verbal immediate memory tasks, and displaying a severe impairment to visual immediate memory.

Assessment of new learning and later recall of verbal information indicated a deficit to learning and recall. P.D. displayed significantly impaired learning of complex verbal information and an inability to recall any of this information, following a 30-min delay. In addition, he displayed a flattened learning curve on serial list learning of unassociated words and impaired retention of these words following an interference trial (RAVLT). It is of interest to note that P.D. displayed intact learning and recall of words on a paired associate task, suggesting that the provision of structure to verbal information aided his learning and recall.

Assessment of P.D.'s ability to learn and recall visual information revealed the presence of deficits, independent of the complexity of stimulus. He displayed an impaired level of immediate and delayed recall for simple facial recognition and an impoverished level of immediate and delayed recall of complex contextual information presented in a visual format. His unprompted recall of a complex geometric design 3 min after having adequately copied it was also impoverished (Fig. 2).

### 2.7. *Language*

P.D. displayed a mild reduction in spoken vocabulary, indicating a relative preservation of word knowledge. Assessment of his ability to name common and uncommon objects also revealed intact ability, consistent with the observation of average word knowledge. Moderate oral dyspraxia was noted in the absence of dysphasic errors.

### 2.8. *Reasoning and concept formation*

P.D. displayed a marked reduction in his ability to understand and make verbal statements. His responses were simplified and concrete but were devoid of dysphasic errors, indicative of reduced verbal conceptual reasoning and not a deficit to language comprehension. In addition, his ability to perform an abstract verbal reasoning task was characterised by a pattern of concrete thinking. Such a pattern of results is indicative of an impairment to reasoning and concept formation, independent of relatively intact language abilities (Lezak, 1995).

### 2.9. *Visuo-spatial orientation*

Across a range of tests involving the use of visuo-spatial abilities, P.D. displayed a pattern of deficits. In the copy trial of the Rey Figure, he performed in a manner, suggestive of impaired planning and strategy formulation but displayed intact visuo-spatial orientation (Fig. 2). However, in the Block Design test, his performances were suggestive of impaired visuo-spatial orientation. On this task, he displayed features of disorientation and a failure to complete designs in a square format. In the Object Assembly test, P.D. displayed correct recognition of the objects, but was

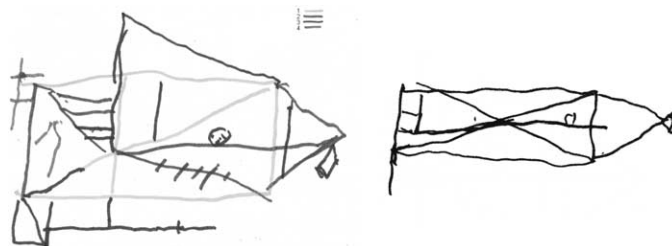


Fig. 2. Complex Figure of Rey Test, copy (left), and recall at 3 min (right).

unable to correctly construct any of the puzzles. The pattern of visuo-spatial deficits apparent on this assessment is consistent with an impairment to visuo-spatial processing manifest as design distortions and impaired constructional strategy use (Lezak, 1995).

#### *2.10. Attention, concentration, and frontal-lobe executive abilities*

Assessment of verbal executive functions revealed a borderline level of abstract concept formation and reasoning. On verbal fluency for letters and category fluency, P.D. performed consistently at an impaired level. On a 24 item Stroop test he displayed a severe impairment in the speed of information processing, evident across all congruent and incongruent conditions. In addition, he displayed a severe impairment to impulse suppression in the incongruent condition.

Assessment of the speed of information processing, sustained complex attention, and concentration revealed a significant impairment to P.D.'s abilities. In the Trail Making Test P.D. displayed a severe impairment to the speed of information processing on both trails and was unable to complete trail B due to an inability to maintain the rules required. In the Colour Form Sort Test, P.D. was able to sort by shape, but was unable to shift to the second category of colour, despite explicit instructions or the presence of an explicit example.

P.D.'s performances on this range of verbal and non-verbal tasks revealed severe impairments to frontal-lobe executive functions manifest as severely impaired attention and concentration, speed of information processing, verbal fluency, impulse suppression, mental flexibility, and abstract reasoning and concept formation.

#### *2.11. Regulation of motor skills*

P.D.'s ability to maintain and regulate motor skills was assessed using the Conflict Knocking Test and Luria's 3-Step Motor Sequence. In the Conflict Knocking Test, P.D. was unable to acquire the conflictual rule with either hand, responding by copying the examiner, despite repetitive explicit instructions in the rule. In the 3-step motor sequence, he was able to copy the 3 steps, but in the absence of an explicit model he displayed bilateral rapid disintegration of the rules to a highly perseverative 2-step sequence.

### **3. Discussion**

P.D. was assessed seven months after sustaining a right thalamic haemorrhage. Examination of his neuropsychological status indicates the presence of the following features: severe anterograde amnesia for verbal and visuo-spatial material; mild retrograde amnesia for episodic information; impaired short-term memory; moderate oral dyspraxia without dysphasia; visuo-spatial deficits; and a pattern of frontal-lobe deficits, including attentional, motor regulation, reasoning and planning deficits, sexual disinhibition, socially inappropriate behaviours, and adynamia and affective blunting. The frontal-lobe deficits evident on neuropsychological assessment were the clinically dominant presentation and are consistent with the notion of a dysexecutive syndrome.

It is pertinent to note that the memory performances exhibited by P.D. were qualitatively different from the memory impairments seen following medial temporal lobe damage. Specifically, P.D. displayed reduced learning of information consistent with poor organisation of information and attention to information as it was received. That he displayed better recognition than recall of information suggests that such information was encoded, but the adequacy of encoding was insufficient to

enable accurate recall. This is further demonstrated by P.D.'s relatively intact learning and recall of paired words, which contrasts strongly with his impoverished learning of prose passages and lists of unrelated words. This preservation of associate learning suggests that P.D. benefited from the structured internal cuing provided by pairing words (Lezak, 1995).

It is also pertinent to note that P.D. did not display any dysphasic disturbance to language function. This suggests that dysphasic disturbances arising from thalamic damage may occur only when damage to the dominant thalamus is implicated. Such inconsistencies between the pattern of deficits evident in P.D. with those of previous case studies of thalamic damage suggest that the notion of a unitary "thalamic syndrome" may be of little clinical relevance.

Damage to the basal forebrain, as evidenced following rupture of an anterior communicating artery aneurysm, has been hypothesised to result in a characteristic pattern of executive deficits. The hallmark features of this anterior communicating artery (AcoA) syndrome have been described as memory problems, confabulation, and executive problems (DeLuca & Diamond, 1995; Beekmans, Vancoillie, & Michiels, 1998; Böttger, Prosiegel, Steiger, & Yassouridis, 1998). However, the presence of confabulation, amnesia, and executive problems is not always evident in cases of AcoA aneurysm (Beekmans et al., 1998). Studies indicate that AcoA aneurysm rupture results in temporally graded retrograde amnesia, intact short-term memory, severe anterograde amnesia, personality changes, and severe executive deficits (Beekmans et al., 1998; Parkin et al., 1994a). It has been suggested that the memory impairments seen in the AcoA syndrome are a consequence of dysexecutive impairment, whereby impaired planning and attentional processes result in the failure to establish adequate retrieval cues at the time of encoding (Parkin, Yeomans, & Bindschaedler, 1994b). Such a dysexecutive memory impairment may explain the pattern of memory and executive deficits exhibited by P.D. following anterior thalamic haemorrhage.

Studies of cases of anterior thalamic haemorrhage and infarction have consistently identified deficits to executive functions, manifest as loss of utilisation behaviour (Hashimoto et al., 1995), impaired sustained and divided attention (Ebert et al., 1999; Hashimoto et al., 1995), impulse inhibition (Hashimoto et al., 1995), impaired abstract reasoning (Ebert et al., 1999), and reduced verbal fluency (Clarke et al., 1994). In addition, personality changes are also regularly noted, following anterior thalamic damage (Clarke et al., 1994). The present case is consistent in finding severe frontal-lobe impairment arising from anterior thalamic damage. While the haemorrhage extended into the right frontal lobe, the presence of verbal executive deficits indicates disruption of left frontal-lobe functions. In addition, the present case demonstrates that lateralised thalamic damage can result in global amnesia. Further, the nature of this amnesia appears to be consistent with a frontal dysexecutive amnesia as opposed to a pure amnesia typically associated with medial temporal lobe damage.

It has been suggested that the amnesia resulting from damage to the thalamus is not solely a consequence of disconnection of the thalamus from the frontal and temporal lobes (Van Der Werf et al., 2000). Rather, it is suggested that the medial thalamus, including anterior nuclei, plays an active role in memory formation (Van Der Werf et al., 2000). While in the present case it is not possible to determine whether or not the mammillo-thalamic tract is affected, the presence of a dysexecutive syndrome and a frontal amnesia suggests a disconnection of the pathways between the frontal lobes and the thalamus is the likely cause. This would contradict the hypothesis that the thalamus is actively involved in memory formation (Van Der Werf et al., 2000) and instead suggests that a disconnection of the anterior thalamus from the frontal lobes is sufficient to explain the neuropsychological deficits noted in this case.



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